

Paediatric Pathology Society

Proceedings of the Twentieth Annual Meeting

The Twentieth Annual Meeting was held in Cardiff on 9 and 10 July 1974, at the University of Wales Medical Teaching Centre.

Scientific communications

Continuing experience of placental villitis of unknown aetiology: harbinger of serious disease? G. Altshuler. Departments of Paediatrics and Pathology, Cincinnati Children's Hospital, and Department of Pathology, Cincinnati General Hospital, University of Cincinnati Medical Center, Ohio.

The recognized causes of chronic intrauterine infections are toxoplasmosis, rubella, cytomegalovirus, herpesvirus, and syphilis. Accumulated experience of the placental pathology of these infections indicates that each causes characteristic morphological abnormalities. In addition the ability to stain spirochaetes and to identify cytomegalovirus inclusions or toxoplasma cysts enables specific diagnoses to be made.

The importance of certain villitis lesions whose aetiology has not been determined has recently been emphasized (Altshuler, 1973). These lesions are morphologically indistinguishable from rubella placentitis. They are focal and are variably proliferative, necrotizing, evanescent, or of reparative, granulomatous appearance. Occasionally an associated basal villitis features lymphoplasmacytic infiltrates. The incidence in our random routine placental examinations is 6%. It is higher in placentas of 'at risk' neonates, whose abnormalities have included prematurity, smallness for gestational age, hepatitis, cataracts, hydrocephalus, myelomeningocele, cardiac malformations, and recurrent reproductive failure. Investigations of over 80 cases, which included many serological and cultural studies, and occasional liver biopsy and necropsy examinations, failed to show that the observed placental villitis was due to any of the above-mentioned causes of chronic intrauterine infection.

REFERENCE

Altshuler, G. (1973). Placental villitis of unknown etiology: harbinger of serious disease? *Journal of Reproductive Medicine*, 11, 215.

Intrauterine *Candida albicans* infection. J. J. van der Harten. Academisch Ziekenhuis, Vrije Universiteit, de Boelelaan 1117, Amsterdam.

Intrauterine *Candida albicans* infection produces characteristic lesions in the placenta and fetal adnexa. Though infections of the maternal genital tract by *Candida* are common in pregnancy, infections of the

fetus by these organisms have only infrequently been reported. This implies that factors other than the mere presence of the fungus favour initiation of placental infection, and may include rupture of the membranes, bleeding, and an intrauterine device (IUD).

Our patient had an IUD, recurring bleeding, but no history of rupture of the membranes. Penetration through intact fetal membranes has, however, been postulated in published reports. The mother lost her IUD between the 13th and 18th week and produced a stillborn fetus in the 23rd week.

Pseudoachondrogenesis and thanatophoric dwarfism. A. J. Barson. Department of Pathology, University of Manchester, Manchester M13 9PL.

True achondroplasia is relatively rare as a cause of dwarfism recognizable at birth and it does not have a high perinatal mortality. There has been confusion in the past between achondroplasia and thanatophoric dwarfism, which is probably the commonest cause of lethal short-limbed dwarfism.

The radiological and pathological features of a case of thanatophoric dwarfism were compared with 2 stillborn sibs originally thought to have achondroplasia. Both sibs had grotesquely shortened and bowed long bones resembling that characteristic of thanatophoric dwarfism, but in addition they had complete absence of mineralization of the vertebral bodies and the sacrum, a feature which is typical of achondrogenesis. These sibs have previously been described as examples of pseudoachondrogenesis (Harris, Patton, and Barson, 1972).

The histopathology of the cartilage of this condition is distinct from thanatophoric dwarfism. In the latter the derangement in maturation of the cartilage cells is seen only in proximity to the costochondral junction of the long bones. In the cases of pseudoachondrogenesis the cartilage cells are immature and swollen in the centres of the epiphysis of the long bones and also within the laryngeal, tracheal, and bronchial cartilage. The differential diagnosis of lethal short-limbed dwarfism is at present primarily made on radiology. There appears also to be histopathological distinguishing features but these are not nearly so well documented.

REFERENCE

Harris, R., Patton, J. T., and Barson, A. J., (1972). Pseudoachondrogenesis with fractures. *Clinical Genetics*, 3, 435.

A familial chromosome abnormality. G. A. Machin. Department of Pathology, Royal Hospital for Sick Children, Sciennes Road, Edinburgh EH9 1LF.